phase in spite of treatment, it is perfectly fair, it seems to me, to advocate it.

I will have to acknowledge that in this patient no test of the uroblin output was made nor of the fragility of the blood cells.

One point interested me, although I do not fully know its value, and that is the Howell-Jolly bodies in the blood. There were relatively few in this instance, and if their presence indicates, as it may, a speeding up of the blood-making function and the calling out of the young cells earlier than normal to fill the ranks in the vessels, their absence would possibly mean a failure at the very point of origin of the red cells—a failure to generate reds rather than a too rapid destruction of them.

SOME UNUSUAL ASPECTS OF EXOPHTHALMIC GOITER.*

By GEORGE D. BARNETT, M. D., San Francisco.
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With the great increase in pathological and experimental work on exophthalmic goiter during the past few years the focus of attention has shifted somewhat from the field of diagnosis to that of pathogenesis, in which our interest has recently been aroused by the stimulating suggestions of Rosenow and Billings. A decade ago the prominent subjects in thyroid literature were early diagnosis, obscure points in diagnosis, formes frustes, etc., yet in spite of the thoroughness with which the question of diagnosis has been exhausted, there is apparently a considerable number of fairly well-marked cases of hyperthyroidism in which the diagnosis is not made, or is greatly obscured by the undue prominence of certain of the less common symptoms. The cases here reported may serve to point out the possibility of such diagnostic error, and to emphasize again the necessity of keeping the thyroid in mind in considering many rather obscure clinical pictures.

Case 1.—Miss F., student of 21 with unimportant family and past history, consulted her family physician in January, 1915, complaining of loss of appetite and malaise. She was found to have a temperature of 103.4; white blood count 9600; Widal negative. Urotropin was given. After a few days began to have frequent burning urination, and blood was discovered in the urine. Temperature rose to 100-103 every afternoon. Tuberculosis of the urinary tract being suspected, the urine was sent to a laboratory for guinea-pig inoculation, and four weeks later the laboratory reported positive tuberculosis. A diagnosis of tuberculosis of the kidney was made, but on account of the absence of any indication of tuberculosis in the ureteral urines, the patient was brought to the hospital in May, 1915, for further investigation.

Physical examination showed a small, well-developed young woman. Thyroid moderately prominent. Marked vasomotor flushing about chest. Pulse 100-124. Systolic blowing murmur at cardiac apex. Slight general abdominal tenderness. Tremor of hands. Knee-jerks lively. Urine 1.011 with trace of albumin and rare hyaline cast. White blood cells 8500: polymorphonuclear 57%, lymphocytes 40%. Hemoglobin 70%. Afternoon temperature 99 to 99.5. Cystoscopic examination showed mild cystitis and some things suggestive of pyelitis. Guinea-pig inoculations negative.

In this case, the rather striking fever at onset naturally occupied the attention of the attending physician, and the attempts to explain it on the basis of kidney infection and to influence that infection by means of hexamethylenamine totally obscured the picture of hyperthyroidism that was doubtless developing during the weeks before the patient came to the hospital. With prolonged rest, overfeeding, hydrobromide of quinine and a discontinuance of bladder therapy there was practically complete relief from symptoms.

Case 2.—Miss W., schoolteacher, complaining of nervousness. Has had five attacks of pneumonia, and has had occasional periods of loss in weight, nervousness and irregular menstruation, but has been able to continue her work. Past history otherwise unimportant. In the summer of 1914 she began to be troubled with nervousness, cardiac palpitation and marked tremor, and lost ten pounds in weight. Physical examination at this time showed slight prominence of eyes, slight enlargement of thyroid; heart rate 120 with systolic blow at apex. A diagnosis of hyperthyroidism was made, and with prolonged rest, quinine hydrobromide, iodine ointment applied over the gland, and a copious non-irritating diet, improvement was marked.

Patient remained practically well up to the middle of January, 1915, when she had an apparent influenza infection for several days, with considerable fever, cough, and some rales in right chest. Was in bed two weeks, the fever continuing, at times as high as 102.5. Pulse 110-120. At the end of this time the temperature fell to normal, the pulse remaining 90 to 100. After a week or so the temperature again rose, and for a period of three weeks reached 100 to 101 each evening. A marked increase in the size of the thyroid was noted with the second rise in temperature. She continued to lose weight, had some sweats, considerable tremor and rapid heart action. White blood count 3700: polymorphonuclears 56%, lymphocytes 36%. Urine normal. X-ray of chest showed nothing positive. Following x-ray treatment of the thyroid, the gland was reduced to about half its former size and became firm, with well-defined borders. Leucopaenia throughout. Widal negative. With continued rest in bed, iodine ointment and quinine hydrobromide in addition to the x-ray therapy, there was gradual improvement. Temperature fell to normal after about three weeks, and remained so except for an occasional transient rise to 99 or 100. Pulse 90 to 110 throughout. Patient left the hospital after four months, weighing more than ever before. No sweats. Eyes practically normal. Thyroid firm and small. No heart murmur. Very little tremor. Has remained well to the present time.

Here, again, without a definite knowledge of the previous attacks, one might well be misled by the striking temperature chart; and of course at the onset of such an attack as the last one the diagnosis must be held in abeyance until all the more usual causes of fever can be excluded.

The question of the frequency and extent of temperature elevation in exophthalmic goiter is one in which there is still difference of opinion. Bertoye, who in 1888 first made a detailed study of the matter, concluded from an analysis of a considerable number of cases that moderate transient fever is of frequent occurrence, and may be found at the onset, during the course of the disease, or only terminally. Kocher, on the other hand, does not consider that fever is a part of the picture in exophthalmic goiter at all, and agrees with Mackenzie that a temperature over 100 is exceedingly rare. In this country, casual mention is made by Barker, and in the papers from the Mayo clinic, of occasional slight fever during the course of the disease, but no emphasis has been laid on its occurrence except by W. Gilman

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Thompson, who, in fifteen, out of a series of forty-three hospital cases reported, found fever of 101 to 104. The fever he describes is "of septic type, oftenest remittent, but sometimes intermittent, always irregular, and occasionally remaining elevated three or four degrees for several consecutive days," in some cases several weeks. It is unaccompanied by change in blood picture or by local manifestations.

Whether the fever is of special significance in exophthalmic goiter, perhaps characterizing a group of cases of different etiology or different degrees of thyroid intoxication, or whether incidental intercurrent infection is to explain the temperature elevation in these cases; or whether, following the work of Rosenow and Billings, we are going to find all exophthalmic goiter to be of infectious origin—these are questions which the near future seems likely to answer. In any event it seems well to point out the possible role of the thyroid in cases of unexplained fever, especially in the absence of leucocytosis.

Cerebral nerve disturbance in exophthalmic goiter, to which attention has recently been directed by Heuer, are among the rarer manifestations of the disease. The following case belongs in this group:

Case 3.—M. D., Danish buttermaker of 33, admitted June 10, 1915, complaining of general weakness and double vision, of six weeks' duration. Family history unimportant. At 21 had swelling in right testicle diagnosed tuberculosis, and testicle was removed. Left testicle was also affected, but healed under open-air treatment. In 1910 had an attack of jaundice lasting three months with vomiting and general weakness but no pain. Recovery following drainage of gall-bladder. In 1912 had repeated attacks of "convulsive vomiting" during a period of two months. Developed very rapid pulse, exopthalmos, and general weakness. Partial thyroidectomy was performed at the San Francisco Hospital, with complete cure, except for slight residual prominence of the eyes. Patient remained well and at work over three years.

About May 15, 1915, six weeks before admission to the hospital, began to notice blurring of vision, followed shortly by persistent double vision. General muscular weakness began about the same time and progressed rapidly. Arms and legs fatigue rapidly, and he has noticed that after chewing a short time he is unable to chew any but the softest foods, and there is a tendency for fluids to run out the nose.

Physical examination showed well-nourished young man. On getting up from supine position lifts head with hands. Moderate exophthalmos. Eye movements limited in all directions. Left eye lags in all movements. Slight ptosis on left. V. Graefe, Moebius, Stellwag present. Distinct weakness of muscles of mastication. Moderate enlargement of left lobe of thyroid. Right lobe apparently removed. No abnormality noted in heart and lungs. Operative scar right rectus. Right testicle gone. Slight fine tremor of extended hands. Tendon reflexes normal. Gait weak, cautious. No spasticity. No ataxia. No Romberg symptom. Stereognosis normal. Some general weakness of skeletal muscles throughout. Deltoids, triceps, scapular muscles, hamstrings and thigh muscles seem to be more affected than the more distal groups. Apparent slight atrophy of supraspinati.

Electrical reactions: No R. D. Both triceps muscles show rapid, though incompelete fatigue to faradic stimulation (interruption 60 per minute). X-ray examination showed normal sella turcica, no evident enlargement of thymus. Urine on one occasion showed trace of sugar and a few finely granular casts. Blood: white cells 5100; polymorphonuclears 51%; lymphocytes 39%. Wassermann negative.

There is here an obvious hyperthyroidism, and in addition many of the symptoms of myasthenia gravis. The relation between these two conditions is an interesting one. Cases of Graves' disease with marked general muscular weakness, with eye muscle paralysis and other bulbar symptoms have been occasionally described for many years; and on the other hand cases of true myasthenia gravis now and then occur with exophthalmos, tachycardia, tremor, and other minor manifestations of hyperthyroidism. Often, as in this case, there is such a confusion of the two pictures that it is difficult to say which is the predominant one. Thus we have here goiter, tremor, exophthalmos, with recovery following operation, and marked eye signs—a striking picture of Graves' disease; and in addition such extensive muscular weakness involving especially the eye muscles and those of mastication and deglutition, with distinct though incomplete faradic fatiguability of both triceps, that a diagnosis of myasthenia gravis seems necessary.

The patient left the hospital, and except for occasional slight remissions, themselves characteristic of myasthenia gravis, became progressively weaker during the following five months. He returned in November, 1915, and died shortly after admission, with acute respiratory failure. Autopsy by Dr. Ophuls showed healed tuberculosis of the lungs; healed tuberculosis of the right kidney (unsuspected during life); healed tuberculosis of testis, with marked atrophy. Thyroid showed marked epithelial proliferation with several areas of round-cell infiltration. Parathyroids not found. No focal lesion in brain tissue.

In this connection it is of interest to recall the possible role of the parathyroids in the etiology of myasthenia. Following the discovery of the parathyroids and the relation between parathyroid insufficiency and tetany there was naturally a search for the condition which parathyroid hyperfunction might be expected to produce. Such a condition was found in the long recognized syndrome of myasthenia gravis, which, as Chvostek remarks, bears the same relation to tetany that the negative does to the picture. And considering the anatomical and functional relationship between thyroid and parathyroids, if myasthenia is hyper-parathyroidism, it is not surprising that it is occasionally associated with hyperthyroidism. However, the objections that have been raised to Chvostek's hypothesis: that in the manifest cases of myasthenia gravis that have come to autopsy, no anatomical evidence of parathyroid hyperfunction has been found; and that successful parathyroid grafts do not produce the slightest evidence of myasthenia in animals—these points must be considered, and leave us again in a state of haziness as to the nature of the condition and the cause of its occasional coincidence with exophthalmic goiter.

It is perhaps worth noting in our case, that

preceding the onset of any of the symptoms there was disease of both testicles necessitating removal of one of them, and producing atrophy of the other. Cases of myasthenia gravis with sexual infantilism have been described. In the present state of our knowledge of the inter-relation of the internal secretions we can only say that we have here a possible initial factor disturbing their equilibrium.

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DISABILITY FROM INJURY TO THE FEET.*

By G. J. McCHESNEY, M. D., San Francisco.

As one of the Medical Referees for the Industrial Accident Commission, I have examined twelve men who have claimed disability from injuries to the feet.

They have exhibited in all sixteen fractures, as follows: Six of one or both malleoli, four of the astragalus, two each of the os calcis and cuneiform, one each of the scaphoid and fifth metatarsal.

Now, this may not seem a series long enough upon which to base any conclusions, but they have been quite instructive to me, and I hope to make them a little so to you.

Their ages varied from twenty-eight to seventy-one, the average being forty-two—men therefore in the prime of life.

The examination was made on an average of eight months after the injury, surely time enough to get a fair estimate of the end results, and these end results were uniformly worse than they should have been.

This is an important point I wish to emphasize; we are not doing our best by this class of injuries. Of course one can say that a referee sees only the bad results, and the good results have no points in dispute requiring his services. This may be so, but there are too many similar results in the practices of all of us, wherein the results should be better than attained at present.

In all the cases seen by me, some ankle-joint motion was always present, was normal in most, and showed little evidence of unskilful treatment. We know that if the astragalus is uninjured, no ankylosis of the ankle-joint is possible, and even if severely fractured, some motion is always possible. This was well shown in one case, where the astragali were badly crushed. The sub-astragaloid joint, however, does not escape so easily. I am convinced that the important role of this joint is lost sight of in over-attention to the more conspicuous ankle-joint above it, which rarely causes trouble. In all these foot injuries seen by me, restriction of sub-astragaloid joint motion furnished most or all of the basis for complaint.

This joint, situated between the os calcis and astragalus, permits a lateral and twisting motion of pronation and supination of the os calcis, which carries the rest of the foot with it through the articulations of the astragalus with the scaphoid and the os calcis with the cuboid. When this joint is fixed by muscle spasm in a pronated position, the so-called rigid flat-foot, or even if a *little* motion is permitted, we have inability to walk upon *rough* ground, *uneven* surfaces, etc., without severe or excruciating pain, as the ability of the foot to accommodate itself to such inequalities is lost. This is not so great a handicap to sedentary city dwellers, walking only upon pavements, but to laborers of all sorts, ladder-climbers, weight-carriers, etc., the disability is well nigh complete. Some, less severely disabled, could walk upon smooth surfaces with no trouble, putting most of the weight squarely upon the heel, but found it impossible to walk comfortably upon uneven surfaces, climb ladders or stairs, do balancing acts, carry weights, etc., thus barring from a status of efficiency all workers except those at benches or desks.

In the malleolar fractures—three Potts, and three fibula alone without fracture of the tarsal bones—the universal mistake had been made of not keeping the proper weight-bearing position of the foot in mind, i. e., slightly supinated or twisted inwards at the sub-astragaloid joint, to throw the weight to the outer side of the foot, where it belongs, and to relax and protect the internal lateral and calcaneo-scaphoid ligaments by holding up the arch. Of course it is not enough to put the foot into this position while the fracture is healing, but in the more crucial period of beginning weight-bearing a high arch-support is very necessary, maintaining this position till the internal lateral ligament and accompanying muscles have shortened and regained their tone, and spasm of the peroneal muscle group has disappeared.

To thus restore the balance of muscles moving the sub-astragaloid joint should not take over two or three months in the simple malleolar fractures, but when the astragalus or os calcis is fractured, unless most skilled and prompt efforts are made to preserve the integrity of this joint, whose importance I have endeavored to set before you, we have a man crippled permanently for all but the slightest occupations.

It has been said that everything in this life is a matter of degree. To fall upon your feet is an expression for good luck, but let us add when the fall is less than ten feet! Over that height it can signify a crippling of the most painful sort.

In the fractures of the os calcis and astragalus—all caused by falls of from ten to thirty feet and landing upon the feet—the predominant symptom was pain and disability referred to the sub-astragaloid joint.

In none of the cases of fractured os calcis or astragalus seen by me, had the treatment been anything more than conservative and expectant. This is wrong. In no case of fracture should more strenuous and repeated efforts be made to secure anatomical restoration of the parts. The average surgeon is usually particular enough to get anatomi-

* Read before the San Francisco County Medical Society, August 15, 1916.